

## Multiple drugs

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**Hypoglobulinaemia, lymphocytopenia and new SARS-CoV-2 virus variant emergence: case report**

A 61-year-old man developed lymphocytopenia during off label treatment with dexamethasone, hypoglobulinaemia during treatment with rituximab and new severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus variant emergence during treatment with remdesivir and off label favipiravir for coronavirus disease 2019 (COVID-19) or follicular lymphoma [*not all routes, dosages and outcomes stated; durations of treatments to reactions onset and outcomes not stated*].

The man was in complete remission of a follicular lymphoma following six cycles of bendamustine and rituximab with an additional two cycles of rituximab finished eight months before COVID-19 diagnosis. He was admitted to a nearby hospital and received favipiravir loading dose 3600mg, and maintenance dose 1600 mg/day and oral dexamethasone. On day 13, he was discharged. He was re-admitted on day 16 and received unspecified antibiotics and oral dexamethasone. On day 19, he developed dyspnea and was transferred to a tertiary hospital. Due to the increased demand for oxygen, he was intubated and had mechanical ventilation on the 2nd day of the third admission. He was managed as the recurrence of COVID-19 infection. He received IV dexamethasone 6.6 mg/day from day 20 for 10 days and remdesivir loading dose 200 mg and maintenance dose: 100 mg/day from day 27 for 10 days. On day 24, he demonstrated hypogammaglobulinaemia (IgG: 229 mg/dL).

The man received immune globulin [immunoglobulin]. He also developed lymphocytopenia from day 20, which continued until day 48 and resolved gradually. On day 116, he was transferred to another hospital for rehabilitation. A total of 13 nasopharyngeal swabs (NPSs) had qRT-PCR, of which ten were positive. The viral load slowly reduced; however, it abruptly increased on day 79. The viral genome was once negative on day 94; however, it was detectable until day 100. NPSs collected on days 20, 49, and 79 revealed cytopathic effects, and the viral propagation was established by qRT-PCR. To examine the genetic trait of the SARS-CoV-2 in a course of infection, near-complete genomes of nine out of ten qRT-PCR positive NPSs were augmented. All seven haplotypes were categorised in 20B in Nextstrain clade or B.1.1.214 in Pangolin Lineage V.3.1.11. When haplotype 1 was compared with Wuhan-Hu-1/2019 (GenBank accession number; MN908947) had ten non-synonymous and five synonymous mutations. Haplotype 2 attained two non-synonymous mutations in two different open reading frames (ORFs) (ORF1b and ORF9b) and one synonymous mutation. One of the non-synonymous mutations, V658I, was situated in nsp12 that encoded RdRp. Haplotype 3 attained additional four non-synonymous mutations, a single deletion (S gene) and a synonymous mutation but it lost two previous non-synonymous mutations including V658I. From haplotype 4 through haplotype 7, diversity was noted majority in the S gene, and two of them (E484Q and S494P) were additional mutations in the receptor-binding motif of the receptor-binding domain (RBD).

Shoji K, et al. Prolonged shedding of infectious viruses with haplotype switches of SARS-CoV-2 in an immunocompromised patient. *Journal of Infection and Chemotherapy* 28: 1001-1004, No. 7, Jul 2022. Available from: URL: <http://doi.org/10.1016/j.jiac.2022.04.004> 803669149